Aggressive periodontitis essay

Sociology, Bullying

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Introduction

Biochemistry is the study of composition, structure and the chemical reactions taking place in living organisms. Dental biochemistry is the study of the composition, structure and chemical reactions taking place in teeth (Chary & Sharma 2004). The teeth form the sensitive part of the human being. The teeth enhance the looks of a human being and it is also for eating. When the dental formula of a human being is correct, the confidence to talk improves unlike when the dental formula is not proper. When care is not taken, the teeth will contact diseases. There are various causes of the diseases in human beings. The health of a human being is an important sphere that every individual must take care of. For the body of the human being to function well, all parts of the body must work well and are in a healthy state (Clerehugh, Tugnait & Genco 2009).

Dental care is essential for a healthy life. Teeth need care for them to last long. Various diseases affect the teeth. Periodontitis is one of the diseases of the teeth. Its clinical manifestations are at an early age. The disease is prevalent in African-American population. It is the infection and the

inflammation of the bones and ligaments that support the teeth in position. Periodontitis falls into three types: aggressive, chronic and necrotizing periodontitis. The tissues supporting the teeth become weak. The primary cause of the periodontitis is bacteria. The genetic factors in individuals also contribute to the aggressive periodontitis. Aggressive periodontitis was referred to as early onset periodontitis (Levine 2011).

Pathogenesis of periodontitis

Aggressive periodontitis is the rapid destruction of the alveolar bone and periodontal ligaments of healthy individuals of a younger age (Reddy 1900). It can lead to loss of teeth at an early age below thirty years if diagnosis and treatment is not done early. The disease manifests itself in generalized and localized form. Several genes are linked to this disease. There is speculation that the familial nature of aggressive periodontitis is the cause of the genetic defect. This gene is responsible for the transmission of the disease (Müller 2005). Aggressive periodontitis is divided into two categories: the localized aggressive periodontitis (LAP) and the generalized aggressive periodontitis (GAP). The aggressive periodontitis is not common like the chronic periodontitis (Armitage 1999).

The rapid destruction of the periodontal causes the generalized aggressive periodontitis. It leads to loss of teeth if not treated appropriately. The disease has racial and sexual elements where the male and black teens being at a high risk of getting infected as compared to females and whites. The disease affects at least three permanent teeth other than first incisors and molars. It has many factors affecting it from environmental, behavioral, genetic, microbiologic and immunologic factors. The pathogenic bacteria in the teeth

especially Porphyromonas gingivitis and Aggregatibacter actinomycetemcomitans have a role in eliciting host response, which determines the immunologic and genetic profile of the patient (Dumitrescu 2010). The environment for example smoking then modifies how the disease will affect the patient. The generalized aggressive periodontitis accompanies a certain collagen enzyme mutations. An example of a generalized form of aggressive periodontitis is the acute necrotizing ulcerative Gingivitis (ANUG) (Dumitrescu 2010). The destruction of the collagen fibers and epithelial attachment occurs with the alveolar bone. The alveolar bone accommodates the teeth.

In human beings, the bone is located in the mandible, which is the lower part of the jaw. It is thick compared to other types of bones to provide support for the teeth and attachment of joints. During the oral activity, the alveolar bone may be exposed. The resultant pain forces the patient to seek medical attention. The plasma membrane in the neurosis raptures and release of inflammatory agents to the outer medium occurs. In the ANUG process, there is release of lysosomal and acid enzymes into the extracellular environment and this activates the acids. The acid chepsin L will hydrolyze the uncalcified collagen fibers. The chepsin L is involved in the digestion process of the antigens. The collagen peptides pass to the blood stream and they provide agents for necrotic bone destruction. This explains the chemical destruction of the teeth in generalized aggressive periodontitis.

Patients complain of progressive spacing of anterior teeth and flaring. The patients also experience bleeding of gums. There is pus discharge and halitosis in the gums. The teeth become mobile later in the infection stages.

There is no severe pain experienced except when infection occurs at tooth apex. Gingival recession occurs and patients experience food impaction because of loss of contact points between the teeth. Patients who smoke face a high risk because their oral hygiene is poor. The disease alternates in a period of quiescence and activity, which leads shows two types during the examination period (Dumitrescu 2010). During the quiescent period, the patients appear free of any symptoms and the gums to be healthy. The gingiva is pink in color. It shows signs of deep periodontal pockets.

The inactivity period may remain for months or years and then there will be signs of active disease. In this period, attachment loss and bone destruction occur. The gingiva shows signs severe inflammation. Gingiva may be tender, edematous, boggy and soft at this stage. Bleeding is evident during this period. In this stage there is inflammation of gingiva. In this stage, the patients opt to seek medical attention. The inflammation may subside and reappear after quiescence period. If left untreated, the advanced stages show mobility and extrusion of teeth. Other patients show loses in weight, general malaise and mental depression (Roshna & Nandakumar 2012). The patients have small amounts of bacterial plague linked to the affected teeth. The amount of the plague in the affected teeth is inconsistent with the amount of the periodontal destruction. The destruction occurs in episodes and it has an advanced period of quiescence as noted earlier. The radiographs show that there is loss of bones in the mouth area.

The localized aggressive periodontitis was formerly called the localized juvenile periodontitis. It starts affecting the patients at adolescent age of between 15 year and 20 years. It shows gingival inflammation, bleeding and

pocketing. The disease affects both the male and the female. The disease localizes mainly to the first incisors or molars with attachment loss. The amount of plague in the affected teeth is minimal and it does not mineralize to form calculus(Roshna & Nandakumar 2012).

The localized aggressive periodontitis contain large amounts negative rod aggregatibacter antinomycetemcomitans(Aa). This bacterium shows two soluble proteins: Leuktosin and cytolethal distending toxin (cdt). A Leukotosin lyses leucocytes that it meets whereas cytolethal distending toxin (cdt) kills the lymphocytes(Roshna & Nandakumar 2012). The maxillary incisors migrate and the diastema formation is affected. In localized aggressive periodontitis, the antibodies binding the toxins appear in blood and facilitate the removal of these toxins after the infection. This prevents the attachment loss from spreading. The antibodies will inhibit the leukotoxin and not the cytolethal distending toxin. There is apparent increase of the clinical crown. The first incisors and molars start to move. During mastication, there is a lot of pain because of the irritation caused on the supporting structures. The teeth become sensitive to any tactile stimuli and thermal features. The regional lymph node may enlarge at this stage. The localized aggressive periodontitis moves very rapidly. The affected teeth lose the alveolar bone in a period of 5 years. The localized distribution occurs after the first permanent teeth, the aggregatibacter actinomycetemcomitans evade the defenses causing periodontal destruction. The localized aggressive periodontitis may relate to a bacterium that is antagonistic to aggregatibacter actinomycetemcomitans hence inhibiting it from further colonizing other periodontal sites in the mouth(Roshna & Nandakumar

2012). Aa may also lose its producing ability because of unknown reasons. When this happens, the disease is arrested and it will not spread to other parts. The other reason is the possibility that the cementum formation is responsible lesion localization. Radiographic findings show vertical loss of the alveolar bone. This occurs around the first incisor and molar in a symmetrical way around the puberty age.

Treatment

The treatment of aggressive periodontitis calls for early diagnosis. This helps in preventing the disease from advancing to other tissues and destruction of the alveolar bones (Hall 2002). Furthermore, because it has chances of familial segregation, examination of the siblings and closely related persons is important. This will help in early diagnosis of the disease. Measures will then be taken to cure it. The treatment of aggressive periodontitis involves rot planning, frequent scaling and adjunctive therapies. The root planning and scaling works at the early stages of the disease while the antibiotic regimens and surgery works at the later stages. When the above methods do not work, extraction is the last option. The therapy should aim at eliminating or suppressing the infectious microorganism. The environment for health teeth is also essential. The other goal is to provide comfort and prevent recurrence of the disease.

The non-surgical therapy is the first anti-microbial therapy. This type manages the disease when it is in its early stages with moderate and mild periodontal symptoms. The therapy starts with attempts of controlling the etiologic agents (Mummolo et. al 2008). The host response plays a role in expression and pathogenesis of the disease. The host response is genetically

determined. The patient himself or the medical team can do the plague control measures. The mechanical method of plague control is by motivating the patient and educating him. The brushing techniques control the plague. The chemical plague control measures include use of chlorohexidine 0. 12%, mouthwashes and iodine (Mummolo et. al 2008). This controls the plaque in the patient. The use of stannous fluoride and Amine fluoride toothpastes and mouth rinses contributes to the mouth hygiene and dental health. The patient should stop smoking because smoking causes aggressive periodontitis. Patients who smoke have more loss of teeth than patients who do not smoke. Non-smokers heal fast compared to smokers

The other method used for treating the aggressive periodontitis is the biological means and the extracellular proteins. A biologic mediator factors like platelet-derived growth and use of extracellular matrix protein give promising results. There are other materials like bone replacement and enamel matrix derivatives (Mummolo et. al 2008).

The biochemistry pertaining to dental disease and health is an important area that helps us understand facts behind the happenings in our dental health. When humans understand the biology and chemistry behind every activity that happens, they will work to prevent and cure the dental diseases (Chary & Sharma2004). The best treatments come from better oral hygiene and every individual should ensure adherence of the standards of oral hygiene. The aggressive periodontitis is curable. This calls for the training of the children to know how to care for their teeth because the periodontitis may affect them if no action is taken. The care of the teeth is essential for a healthy nation. The funds used for curing the dental patients could be used

to develop other sectors of the economy. Conclusion

The prevalence of aggressive periodontitis is low but its management is very challenging because of the genetic predisposition. The genetic factor is not modifiable but prevention remains the better option. The etiology understanding, pathogenesis, host immune response and risk factors are also an important aspect in preventing the aggressive periodontitis. The advances in tissues engineering, regenerative concepts and gene therapy is also an important factor that the dental patients need to know.

References

Armitage G. C., 1999. Development of a classification system for periodontal diseases and conditions. Annals of Periodontology / the American Academy of Periodontology. 4, pp. 1-6.

Chary, T. M., & Sharma, H., 2004. Practical biochemistry for medical and dental students. New Delhi, Jaypee Brothers.

Clerehugh, V., Tugnait, A., & Genco, R. J., 2009. Periodontology at a glance. Chichester, U. K., Wiley-Blackwell.

Dumitrescu, A. L., 2010. Etiology and pathogenesis of periodontal disease. Heidelberg, Springer.

Hall, W. B., 2002. Critical decisions in periodontology. Hamilton, Ont, B C Decker.

Levine, M., 2011. Topics in dental biochemistry. Berlin, Springer.

Mummolo, S., Marchetti, E., Di Martino, S., Scorzetti, L., & Marzo, G., 2008. Aggressive Periodontitis: Laser Nd: YAG treatment versus conventional surgical therapy. European Journal of Paediatric Dentistry, 9(2), pp. 88–92.

Müller, H. P., (2005). Periodontology: the essentials. Stuttgart, Thieme.

Reddy, S., 1900. Essentials of clinical periodontology and periodontics. New Delhi, Jaypeed Bros. Medical Publishers.

Roshna, T. & Nandakumar, K. 2012. Generalized Aggressive Periodontitis and Its Treatment Options: Case Reports and Review of the Literature. Case Reports in Medicine. http://dx. doi. org/10. 1155/2012/535321.